Social context and sex moderate the association between Type D personality and cardiovascular reactivity

Adam O' Riordan[1*], Siobhán Howard[1,2], Stephen Gallagher[1,2]

[1]Department of Psychology, Centre for Social Issues Research, Study of Anxiety, Stress and Health Laboratory, University of Limerick, Limerick, Ireland

[2]Health Research Institute, University of Limerick, Ireland

*Corresponding author

Adam O' Riordan

E-mail address: adam.oriordan@ul.ie

Ph: + 353 (0) 61 234899

Fax: +353 (0) 61 233211

Abstract

Background: Type D personality has been consistently associated with adverse cardiovascular health with atypical cardiovascular reactions to psychological stress one plausible underlying mechanism. However, whether this varies by sex and social context has received little attention. **Purpose:** This study examined the interaction between Type D personality, sex and social context on cardiovascular reactivity to acute stress.

Methods: A sample of 76 healthy undergraduate students (47 female) completed the DS14 Type D measure, before undergoing a traditional cardiovascular reactivity protocol. The social context of the laboratory environment was manipulated to create a social and non-social context using a between-subjects design. Systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR) were monitored throughout. **Results:** No associations were evident for blood pressure. However, a significant personality × sex × social context interaction on HR reactivity was found; here Type D was associated with a higher HR response to the social task amongst males but not females, while Type D females typically exhibited blunted reactions. **Conclusions:** While these atypical reactions indicate a possible psychophysiological pathway leading to adverse cardiovascular events amongst Type Ds, it appears that Type D males are particularly vulnerable to socially based stressors, exhibiting exaggerated cardiovascular reactions.

Key Words: Type D personality, cardiovascular reactivity, stress, biological sex, social context

1. Introduction

Type D personality, characterised by high levels of both negative affectivity (the tendency to experience negative emotions) and social inhibition (the tendency to inhibit the expression of emotions in social situations) has been associated with adverse health-related outcomes, primarily in cardiac populations. The combination of both negative affectivity and social inhibition has been linked to poor prognosis among cardiac patients (Denollet et al. 2006; Denollet et al. 2000; Martens et al. 2010; Schiffer et al. 2008), as well as to both cardiac and all-cause mortality (Denollet et al. 2006; Denollet et al. 2000; Martens et al. 2010; Denollet et al. 1996; Schiffer et al. 2010). Although a smaller number of studies have reported null findings (Coyne et al. 2011; Pelle et al. 2010; Meyer et al. 2014; Grande et al. 2011), a meta-analysis has found Type D personality to predict a 2-fold increased risk of cardiac events (Grande et al. 2012).

While a link between Type D personality and cardiovascular disease has been established, the precise mechanistic pathway underpinning this relationship remains unclear. Evidence suggests that this may be facilitated via indirect mechanisms, such as poor adherence to medical treatments and engagement in adverse health-related behaviours (Williams et al. 2011; Williams et al. 2008). Direct mechanisms via psychophysiological processes, such as cardiovascular reactivity to stress, have also received substantial support (Howard et al. 2011; Kelly-Hughes et al. 2014; Kupper et al. 2013b; O'Leary et al. 2013; Williams et al. 2009).

The reactivity hypothesis posits that exaggerated or prolonged cardiovascular responses to psychological stress promotes the development of cardiovascular disease (Obrist 1981). This hypothesis has received considerable support with prospective studies identifying that exaggerated cardiovascular reactivity predicts hypertension (Carroll et al. 2012b; Markovitz et al. 1998), atherosclerosis (Matthews et al. 1998; Barnett et al. 1997), increased left ventricular mass (Georgiades et al. 1997; Murdison et al. 1998), and cardiovascular disease mortality (Carroll et al. 2012b). Although low cardiovascular reactivity to acute stress is often assumed to be benign, prior evidence also indicates that diminished or ‘blunted’ cardiovascular responses to stress are also associated with negative health states, including obesity (Carroll et al. 2008), poor cognitive functioning (Ginty et al. 2012), increased intima-media thickness (Ginty et al. 2016), and all-cause mortality among heart failure patients (Kupper et al. 2015). Thus, it has now been posited that both exaggerated and blunted cardiovascular responses to stress imply a homeostatic dysfunction and psychosomatic disease vulnerability (Lovallo 2011). Moreover, it is worth noting that Type D personality has been associated with both exaggerated (Kupper et al. 2013b; Williams et al. 2009), and blunted (Howard et al. 2011; O'Leary et al. 2013; Kelly-Hughes et al. 2014; Kupper et al. 2013a) cardiovascular responses to stress.

One notable and likely explanation for these differential cardiovascular responses exhibited by Type Ds across studies pertain to the social salience of the experimentally manipulated stressor. The socially inhibited facet of Type D personality encapsulates feelings of insecurity and discomfort when in the presence of others (Sher 2005) and previous research suggests that Type D personality is characterised by a cognitive bias towards interpreting social situations as distressing (Grynberg et al. 2012; Howard et al. 2018). Thus, it is likely that Type D individuals are more vulnerable to social rather than non-social stressors. Studies that have solely employed non-social stress tasks (e.g. mental arithmetic and multitasking stressors) have predominately found Type D individuals to exhibit blunted cardiovascular reactions (Howard et al. 2011; Kelly-Hughes et al. 2014). These studies have identified that Type D women in particular, show blunted cardiovascular reactions. In contrast, it appears that social stressors elicit an exaggerated cardiovascular response. For example, prior research that has compared the reactions of Type D and non-Type D individuals to both social and non-social stressors have found Type D individuals to exhibit exaggerated reactions to a socially evaluative stressor, but not to an analogous non-social stressor (Bibbey et al. 2015). Similarly, Type Ds have been found to exhibit an exaggerated cardiovascular response to a cold pressor task whilst being observed (Kupper et al. 2013b); it is possible that the monitoring of the participants heightened the socially evaluative context of the laboratory. This exaggerated cardiovascular response exhibited by Type D individuals during socially salient conditions is likely to be explained by their socially inhibited nature, whereby they find social contexts more stressful in comparison to non-social contexts. Indeed, support for this notion comes from studies that show the social inhibition subcomponent of Type D personality to be associated with exaggerated blood pressure reactivity in response to a social salient stressor (Habra et al. 2003). Although these task-related findings are not consistent across all studies (O'Leary et al. 2013; Williams et al. 2009), it does appear that Type D individuals primarily exhibit exaggerated cardiovascular responses to social tasks, and blunted cardiovascular responses to non-social tasks.

In addition to the social salience of the stress task, sex has also been proposed as an influential factor regarding Type D personality and cardiovascular reactivity, with adverse cardiovascular responses exhibited primarily by Type D males (Howard and Hughes 2013; Kupper et al. 2013b; Williams et al. 2009). Moreover, whilst some studies have found Type D females to exhibit blunted cardiovascular responses (Howard et al. 2011; O'Leary et al. 2013), Type D males have been noted to exhibit exaggerated cardiovascular reactions (Williams et al. 2009; Kupper et al. 2013b), to initial stress and maladaptive patterns of habituation to reoccurring stress (Howard and Hughes 2013). It has been posited that social norms pertaining to how one self-presents interpersonally differs between males and females, and the propensity to behave in a socially inhibited manner is less acceptable and thus more stressful for males, especially during interpersonal challenges that require social expression

(Habra et al. 2003). Similarly, it has been argued that the tendency to inhibit the expression of emotions is a sex-stereotyped behaviour and the fundamental element of Type D personality engendering differential cardiovascular responses in Type D males and females (Howard and Hughes 2013). This is plausible given that social inhibition has been associated with exaggerated cardiovascular reactivity to socially salient stressors among males but not females (Habra et al. 2003). Thus, it is likely that Type Ds, and in particular Type D males, are more vulnerable to socially based stressors.

Whilst some have noted a relationship between Type D personality and cardiovascular reactivity when solely utilizing the tradition dichotomous Type D construct (Williams et al. 2009; Bibbey et al. 2015), evidence suggests that Type D personality is better represented as a continuous construct based on the product of the social inhibition and negative affect subscales (Ferguson et al. 2009). However, additional studies have confirmed the association between Type D and cardiovascular reactivity when using the continuous (Kelly-Hughes et al. 2014) or both constructs (Kupper et al. 2013b; Kupper et al. 2013a; O'Leary et al. 2013; Howard et al. 2011)

Therefore, the primary aim of the present study is to extend the findings of the Bibbey et al. (2015) study, by examining how *both* sex and the social context of the experimental stressor moderate the relationship between Type D personality and cardiovascular reactivity. Unlike previous research manipulating the social conditions of the task, we used precisely the same tasks, procedure, and instructions for participants in both experimental groups; instead, we used the order of the tasks as a manipulation of the social conditions in the laboratory. By using both a mental arithmetic task and a speech task, we were able to examine reactivity to both stressors combined and identify if completing the speech or the mental arithmetic task first resulted in different patterns of cardiovascular reactivity between Type D and non-Type D individuals. Given Type D’s propensity towards a cognitive interpretation bias in ambiguous social situations (Grynberg et al. 2012), it was proposed that completing the speech task rather than the mental arithmetic first would heighten the social context of the laboratory environment.

2. Materials and methods

2.1 Design

The study employed a 2 × 2 between subjects factorial design with Type D examined as covariate. The independent variables were sex (males and females), social context (social and non-social), and Type D personality. Type D was computed as the product of the SI and NA raw scores (Howard and Hughes 2013; Howard et al. 2011; Howard et al. 2018). The dependent variables included systolic blood pressure (SBP), diastolic blood pressure (DBP) and heart rate (HR) reactivity. Reactivity scores were computed as the difference between mean baseline and mean task value for each cardiovascular parameter (Phillips et al. 2009; Gallagher et al. 2018).

2.2 Participants

Seventy-six healthy undergraduate students (61.8% female) participated in this study. All participants were recruited using the university's online research participation system and were provided with course credits in exchange for their participation. Participants ranged in age from 18-58 years (M = 21.99, SD = 6.33) with a mean body mass index (BMI) of 24.17 kg/m^2 (SD = 3.95). While 40 participants were assigned to the non-social condition (26 female), 36 were assigned to the social condition (21 female). The sample size used in the current study is of similar magnitude to analogous studies examining two and three way interactions with the Type D construct (O'Leary et al. 2013; Howard and Hughes 2013;

Howard et al. 2011). In order to minimise the influence of confounding variables, participants with a diagnosis of cardiovascular disease, hypertension, or an immune disorder were excluded from the study. Further, due to the subsequent alteration in blood pressure following smoking (Cruickshank et al. 1989; James and Richardson 1991) and caffeine consumption (Hartley et al. 2000; Savoca et al. 2005), participants were asked to refrain from smoking and caffeine intake for 2 hours before the laboratory session. In addition, to avoid any potential impact of alcohol intake (Potter et al. 1986) and exercise (Somers et al. 1991) on subsequent blood pressure, participants were asked to refrain from vigorous exercise and alcohol for 12 hours prior to testing.

2.3 Measures

2.3.1 Type D Measure

The DS14 (Denollet 2005), a 14-item scale measuring both social inhibition (SI; 7 items) and negative affectivity (NA; 7 items) was used as a measure of Type D personality. Participants respond on a 5-point Likert scale ranging from 0 = false to 5 = true to a range of statements measuring both SI and NA. Examples of items measuring SI are ‘I often feel inhibited in social interactions’ and ‘I am a closed kind of person’, while NA is assessed using items such as ‘I often feel unhappy’ and ‘I am often irritated’. Scores on each subscale can range from 0-28. The overall DS14 has been found to be internally consistent, with Cronbach’s alpha > .86 reported for both subscales (Denollet 2005). In the present study, the Cronbach’s α was .79 and .79 for SI and NA subscales respectively, indicative of high internal consistency. Prior research suggests that Type D personality should be represented as a dimensional rather than a categorical construct (Ferguson et al. 2009), and therefore the continuous Type D construct was computed as the product of the SI and NA subscales (Howard and Hughes 2013; Howard et al. 2011)

2.3.2 Cardiovascular Measurement

Continuous non-invasive measurements of SBP, DBP and HR were recorded using a Finometer Pro hemodynamic cardiovascular monitor (Finapres Medical Systems BV, BT Arnhem, The Netherlands). The Finometer takes continuous non-invasive measurements from the finger arterial pressure, based on the volume clamp method initially developed by Penaz (1973). Along with the finger cuff, an arm cuff is used to calibrate reconstructions of the intra-brachial pressure obtained from the finger. The Finometer uses a hydrostatic height correction system is order to correct participant's hand height to heart level. The Finometer has been extensively used in prior cardiovascular psychophysiological research (Gallagher et al. 2018; Howard et al. 2011) and has been noted to provide an accurate measure of blood pressure (Guelen et al. 2003; A. Schutte et al. 2004; A. E. Schutte et al. 2003).

2.3.3 Stress Task Measures

Immediately before and after the stress task participants were provided with a self-report rating scale, assessing how stressful they *expected* to find the task and how stressful they subsequently *found* the task. Participants were asked to respond to both items on a 7-point Likert scale (0= Not at all, 6= Extremely). These measures were used to determine if the stress task was perceived as psychologically stressful and were adapted from previously used psychological stress task questionnaires (Gallagher et al. 2014; Phillips et al. 2009; Gallagher et al. 2018).

2.3.4 Demographic and Anthropometric Variables

A stadiometer and weighing scales were used to measure height and weight. Socio-demographic information including smoking status, age, nationality, sex and marital status were assessed using a standardised demographic questionnaire.

2.4 Stress Task

Two stress-tasks were used in the present study; a serial subtraction task and a speech task. The paced auditory serial addition test (PASAT) (Gronwall 1977) was used as it has been found to successfully perturb the cardiovascular system (Gallagher et al. 2014; Phillips et al. 2009). During this task, participants were auditorily presented with single digit numbers via a laptop and speaker. Participants were required to retain each digit and add it to the subsequent digit presented, returning the answer verbally. The speed at which the digits were presented increased throughout the four-minute task, thereby increasing the cognitive demand on the participant. All instructions were provided to the participants via a pre-recorded video on the laptop and the researcher provided no feedback to participants during the task, completely eliminating all communication. Furthermore, the researcher also sat behind an opaque screen; eliminating any interaction between researcher and participant.

During the speech task, participants were required to prepare a four-minute speech in which there were instructed to describe three of their best and worst characteristics, with the use of real life examples (Bosch et al. 2009). Participants were given 2 minutes to prepare their speech. The researcher remained behind an opaque screen but ensured consistent direct communication by continually instructing the participant to continue speaking the moment they ceased. Thus, the speech task was designed to implement elements of direct and reciprocal communication accompanied by the requirement to continually speak, engendering a more socially orientated task. Together, these two tasks offered comparable actions on behalf of the participant (communitive speech).

The first stress task completed by participants during experimentally manipulated stress conditions has been noted to determine their physiological (cardiovascular and endocrine) responses to the overall stress protocol (Al'Absi et al. 1997; Linden et al. 1985). Thus, we experimentally manipulated the social salience of the stress conditions by presenting either the speech or the PASAT (mental arithmetic) first; in this way, we manipulated the social conditions of the tasks, while using the same tasks in both experimental groups. While all participants completed the same tasks, participants who undertook the speech task first completed the tasks under socially salient conditions; participants who completed the PASAT first had the social salience of the conditions minimised. The tasks were completed in succession with no recovery period between. A random number generator using an excel formula was used to randomly assign each participant to either the social or non-social condition.

2.5 Procedure

All testing took place at the health and psychophysiology laboratory at our University. Upon arrival, participants were greeted by the researcher who went through a brief checklist of exclusionary criterion. From the moment of arrival, participants were allocated a 20-minute period to acclimatise to the laboratory setting. During this time participants read the information sheet, signed the consent form and had their height and weight measured for calculation of body mass index (BMI). Participants were seated at a desk on which a laptop and table lamp were placed. In order to control for movements that could potentially influence the assessment of cardiovascular parameters, participants were instructed to place their feet into a basin located under the desk. Participants were then provided with a demographic questionnaire. Subsequently, resting cardiovascular parameters were assessed for 10 minutes. In order to obtain a ‘vanilla’ baseline (Jennings et al. 1992), participants continued to complete psychometric questionnaires and were provided with reading material during this period. Immediately before the stress task began, participants completed the pre-stress task questionnaire and the experimenter then switched off the main lights in the laboratory. They completed the stress task under the spotlight of the table lamp. Further, the researcher wore a white laboratory coat throughout the experimental procedure. These conditions were manufactured in order to augment stress levels and ensure a psychological separation between the researcher and participants. The presentation of the PASAT or speech task first was counterbalanced across the procedure as an experimental manipulation of the social context of the laboratory. Following the stress-task the main lights were switched back on, and participants completed the post-task questionnaire and DS14. The Finometer cuff was then detached and participants were provided with a debriefing sheet, thanked for their participation and then left the laboratory.

2.6 Data analyses

Change scores were computed by subtracting mean task values computed from both tasks, from mean baseline values, thereby returning a delta score indicative of reactivity. A preliminary analysis was conducted in order identify outliers and cardiovascular reactivity scores deviating +/- 3.00 SD from the mean were considered outliers and excluded from analyses; one outlier on SBP reactivity was identified. Further, three participants were missing baseline and/or stress task cardiovascular measures and thus, reactivity scores for these participants could not be computed. In order to determine if the stress protocol was successful in perturbing the cardiovascular system, a series of simple paired-samples *t*-tests (baseline, task) were conducted on each cardiovascular parameter. Similarly, in order to determine if the stress protocol was psychologically stressful, a paired-samples *t*-test (pre and post task) was conducted on self-reported stress. Pearson's correlations were used to test the association between Type D and resting cardiovascular parameters. In order to test the main hypothesis a series of 2 (sex: male vs. female) × 2 (social context: non-social vs. social) × 1 (continuous Type D construct) ANCOVAs were conducted, with Type D entered into the model as a covariate. Identification of significant interactions with the covariate (Type D) is possible by examining a custom-built model aimed to test the homogeneity of regression slopes function (Howard and Hughes 2013; O'Leary et al. 2013). In order to control for potential confounding variables, BMI, smoking status, age and baseline cardiovascular measures were entered into the model as covariates.

3. Results

3.1 Manipulation check and Preliminary Analyses

A series of paired-samples t-tests revealed that the stress task successfully perturbed cardiovascular activity: for SBP, $t(72) = 12.5$, $p < .001$, for DBP, $t(72) = 14.99$, $p < .001$ and for HR, $t(72) = 8.54$, $p < .001$. As can be seen in Table 1, scores on SBP ($M = 142.21$, $SD = 16.47$), DBP ($M = 86.16$, $SD = 9.88$), and HR ($M = 85.15$, $SD = 12.28$) were significantly higher during the task compared to baseline SBP ($M = 124.68$, $SD = 11.32$), DBP ($M = 75.5$, $SD = 7.57$) and HR ($M = 79.79$, $SD = 10.52$). Descriptive statistics for reactivity variables are displayed in Table 2. Similarly, a paired sample t-test confirmed a significant increase in pre-task ($M = 3.23$, $SD = 1.41$) to post-task ($M = 4.29$, $SD = 1.36$) rating of self-reported stress, $t(75) = 7.26$, $p < .001$, indicating that the stress protocol was also perceived as psychologically stressful.

There was no significant association between Type D personality and resting SBP, $r = -.04$, $p = .73$, or DBP, $r = +.11$, $p = .35$. However, Type D personality was associated with increased resting HR, $r = +.33$, $p = .01$. A follow-up analysis revealed a significant association between Type D personality and resting HR for males, $r = +.41$, $p = .03$, but not for females $r = +.21$, $p = .16$.

[Insert Table 1 About Here]

3.2 Type D Personality and Cardiovascular Reactivity to Stress

There was no significant main effect of Type D personality on SBP, $F(1, 60) = .04$, $p = .835$, $\eta^2_p = .001$, DBP, $F(1, 61) = .05$, $p = .832$, $\eta^2_p = .001$, or HR reactivity, $F(1, 61) = .38$, $p = .541$ $\eta^2_p = .01$. However, there was a significant sex × personality interaction on HR reactivity, $F(1, 61) = 5.10$, $p = .027$, $\eta^2_p = .08$, with continuous Type D scores inversely associated with HR reactivity in females, with no association between Type D scores and HR reactivity in males.

There was also a significant social context × personality interaction on HR reactivity, $F(1, 61) = 5.76$, $p = .019$, $\eta^2_p = .09$, indicating that the relationship between Type D personality and HR reactivity varied across the social context of the laboratory situation. Here, Type D personality was positively associated with HR reactivity to the social conditions and inversely associated with HR reactivity to the non-social conditions. However, these two-way interaction effects were qualified by a significant sex × context × personality interaction on HR reactivity, $F(1, 61) = 4.52$, $p = .038$, $\eta^2_p = .07$. Follow up analyses yielded a significant personality × sex interaction on HR reactivity to the social condition, $F(1, 25) = 10.89$, $p = .003$, $\eta^2_p = .30$, but not to the non-social condition $F(1, 31) = .45$, $p = .507$, $\eta^2_p = .01$. As illustrated in Figure 1, while Type D women had a lower HR response to the social condition, male Type Ds exhibited exaggerated HR reactions.

There was no significant main effect of personality on HR reactivity to either the maths task, $F(1, 63) = 1.47$, $p = .23$, $\eta^2_p = .02$, or the speech task, $F(1, 63) = .31$, $p = .579$, $\eta^2_p = .01$, when examined separately. Similarly, there was no significant sex × personality interaction on HR reactivity to either the maths task $F(1, 63) = .29$, $p = .593$, $\eta^2_p = .005$ or the speech task $F(1, 63) = .33$, $p = .571$, $\eta^2_p = .01$. These null findings revealed that the significant effects on HR reactivity were not driven by either task alone, but by the combination of both tasks. Finally, there were no main or interaction effects for Type D personality, social context or sex on either SBP or DBP reactivity.

4. Discussion

The present study sought to elucidate the relationship between Type D personality and cardiovascular reactivity to stress under both a non-social and social condition, using the same stress-tasks with a subtle manipulation of laboratory conditions to either enhance or limit the social salience of the lab environment. Further, we sought to examine if sex moderated the relationship between Type D personality and cardiovascular reactivity, given previous research that suggests Type D may be particularly damaging to males. Overall, we found that subtle manipulations of the social conditions in the laboratory resulted in different patterns of HR reactivity to the same stressors; an effect moderated by sex. As hypothesised, Type D men are more physiologically vulnerable to subtle social conditions, showing elevated cardiovascular responses. This is consistent with previous findings demonstrating that Type D personality is associated with exaggerated cardiovascular reactivity amongst males (Williams et al. 2009) and in response to socially evaluative stressors (Bibbey et al. 2015). On the contrary, Type D women show lower cardiovascular reactions to the overall stress task, regardless of the social condition.

Prior studies examining the relationship between Type D personality and cardiovascular reactivity have yielded mixed results, with Type D associated with both blunted (Howard et al. 2011; Kelly-Hughes et al. 2014; Kupper et al. 2013b; O'Leary et al. 2013; Kupper et al. 2013a) and exaggerated cardiovascular responses (Kupper et al. 2013b; Williams et al. 2009) to stress. We argued that two salient features may explain these disparate findings; social nature of the experimental stress task and sex. Type D females exhibited blunted reactions to the stress task, irrespective of the social context. Furthermore, Type D personality was associated with exaggerated reactivity to the social condition, particularly amongst males. Thus, sex moderated the relationship between Type D personality and cardiovascular reactivity to the social and non-social condition differently.

Furthermore, we found sex significantly moderated the relationship between Type D personality and cardiovascular reactivity to the social but not the non-social conditions. During the social condition, Type D personality was associated with an exaggerated HR reactivity amongst males, but not females. This is consistent with previous research indicating that the relationship between Type D personality and cardiovascular reactivity is particularly adverse among males. Previously, Type D personality has been associated with exaggerated cardiac output (Williams et al. 2009) and heart period reactivity (Kupper et al. 2013b) amongst males, but not females. Similarly, in response to recurrent stress, Type D males have been noted to display cardiovascular sensitisation, in which their cardiovascular responses increased from the first to the second exposure to the stressor (Howard and Hughes 2013). Together, evidence points towards the Type D personality construct as particularly damaging to males. It is postulated that social norms for men and women differ regarding how they self-present interpersonally, and the propensity to behave in a socially inhibited manner is less acceptable and therefore more stressful for males, especially during interpersonal challenges that require social expression (Habra et al. 2003). Thus, it is likely that Type Ds and in particular, Type D males are specifically vulnerable to socially based stressors, possibly accounting for the exaggerated cardiovascular responses exhibited by Type D males during the social condition. The majority of research on which Type D personality has been shown to predict negative outcomes following a cardiac event are based on samples of predominantly male participants (Denollet et al. 2006; Denollet et al. 2000; Schiffer et al. 2008). The interaction found in the current study suggests that exaggerated cardiovascular reactivity to social stressors may be an important pathway leading to negative cardiovascular health for Type D males. Additionally, the present study also confirms previous findings identifying a reduced HR response in Type D females. Previously, Type D women have been found to exhibit lower HR reactivity to a mental arithmetic stressor (Howard et al. 2011).

Previously, the social context of the task was highlighted as an important moderator of the stress response in Type D and non-Type D individuals (Bibbey et al. 2015). However, Bibbey et al. (2015) 1) did not examine whether sex moderated this effect, and 2) used "extreme" cases of Type D, choosing individuals at the upper and lower end of the Type D scale quartiles. The present study extends the findings of Bibbey et al. (2015) by identifying that Type D males may be particularly vulnerable to these socially based stressors, using the normally distributed continuous Type D variable rather than the categorical Type D classification. However, it is worth nothing that our findings for HR is somewhat different, as several other studies have found an effect of Type D on blood pressure (Bibbey et al. 2015; Kelly-Hughes et al. 2014; Kupper et al. 2013a; Kupper et al. 2013b; O'Leary et al. 2013)

While other studies have found main effects for blood pressure, when they examined the interaction with sex similar findings to ours are observed i.e., an exaggerated HR response in males was found. Moreover, given that cardiac output is a product of HR, it is worth noting that Type D males were previously found to have an increased cardiac output reactivity to stress (Williams et al. 2009). The results of the current study corroborate these findings, with Type D personality associated with increased resting HR and exaggerated HR reactivity amongst males. Furthermore, given the activation of the autonomic nervous system and hypothalamic-pituitary-adrenal axis during exposure to acute psychological stressors (Steptoe and Brydon 2009), the findings from the current study indicate that Type D personality amongst males may be characterised by a chronic dysfunction of the hypothalamic-pituitary-adrenal axis and a shift in autonomic balance towards sympathetic rather than parasympathetic activation. This is a highly plausible postulate considering that sympathetic stimulation is central to increased heart rate and myocardial contractility, resulting in increased cardiac output (Gordan et al. 2015). In addition, several prospective studies have linked exaggerated cardiovascular reactivity and elevated resting heart rate to adverse cardiovascular health (Carroll et al. 2012b; Markovitz et al. 1998; Carroll et al. 2012a; Wang et al. 2014; Tadic et al. 2018), indicating a potential mechanistic underpinning facilitating the relationship between Type D personality and negative cardiovascular outcomes.

One of the strengths of the present study is the subtle manipulation of the social context of the laboratory, but using precisely the same tasks in the computation of reactivity. By experimentally manipulating the social salience of the stress task by either presenting the mental arithmetic or speech task first, we were able to show that the social nature of the laboratory environment resulted in altered patterns of stress responding in Type D individuals. This experimental manipulation was based on prior evidence suggesting that Type D individuals have a cognitive interpretation bias where they view ambiguously neutral social situations as more distressing (Grynberg et al. 2012). Our study attempted to employ this interpretation bias in its experimental manipulation of the social conditions in the lab.

The findings are limited by the relatively small sample size, although it is of a similar magnitude to analogous studies examining two and three way interactions with the Type D construct (O'Leary et al. 2013; Howard and Hughes 2013; Howard et al. 2011). While we attempted to recruit equal numbers of males and females to the study, we still failed to recruit equal number of males, with our sample comprising 62% females. The results from this study and others strongly suggest that sex is an important moderating variable to consider in Type D-reactivity studies. Furthermore, the sample consisted of undergraduate students mainly of a relatively young age, with specific sample characteristics, thus it is questionable if the results are generalizable to other cohorts. Finally, differences in reactivity may reflect physical differences in task dimensions (e.g. speaking speed), whereby Type D individuals exhibit physical differences due to their socially inhibited nature when performing speech tasks. Thus, future research should employ speech tasks with greater standardization regarding the quantum of task-load, whereby physiological reactions cannot be attributable to physical differences in task dimensions. Additionally, we recommend for future research to include self-report questions to assess perceptions of social evaluation during the stress task.

One strength of using healthy samples rather than clinical samples is that it avoids potential confounds associated with having existing disease. Nevertheless, we recommend that future studies recruit different cohorts to confirm the association between Type D personality, sex and cardiovascular reactivity to non-social and social stressors.

In sum, the current study extends existing research by examining sex as a moderator of the relationship between Type D personality and cardiovascular reactivity to stressors of different social saliences. Firstly, the current study found that the effect of Type D personality on cardiovascular reactivity varied across the social conditions of the laboratory environment. Secondly, it appears that Type D individuals are particularly vulnerable to socially salient stressors, exhibiting greater cardiovascular responses. Finally, we found that sex moderates the relationship between Type D personality and cardiovascular reactivity to stress under social conditions, with Type D males particularly vulnerable to socially based stressors, indicating a potential psychophysiological pathway leading to adverse cardiovascular events amongst Type D men.

Compliance with Ethical Standards

Conflict-of-Interest Statement: The authors declare that they have no conflict of interest.

Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent: Informed consent was obtained from all individual participants included in the study

References

Al'Absi, M., Bongard, S., Buchanan, T., Pincomb, G. A., Licinio, J., & Lovallo, W. R. (1997). Cardiovascular and neuroendocrine adjustment to public speaking and mental arithmetic stressors. *Psychophysiology, 34*(3), 266-275.

Barnett, P. A., Spence, J. D., Manuck, S. B., & Jennings, J. R. (1997). Psychological stress and the progression of carotid artery disease. *Journal of hypertension, 15*(1), 49-55.

Bibbey, A., Carroll, D., Ginty, A. T., & Phillips, A. C. (2015). Cardiovascular and cortisol reactions to acute psychological stress under conditions of high versus low social evaluative threat: associations with the type D personality construct. *Psychosomatic Medicine, 77*(5), 599-608.

Bosch, J. A., De Geus, E. J., Carroll, D., Goedhart, A. D., Anane, L. A., van Zanten, J. J. V., et al. (2009). A general enhancement of autonomic and cortisol responses during social evaluative threat. *Psychosomatic Medicine, 71*(8), 877.

Carroll, D., Ginty, A. T., Der, G., Hunt, K., Benzeval, M., & Phillips, A. C. (2012a). Increased blood pressure reactions to acute mental stress are associated with 16-year cardiovascular disease mortality. *Psychophysiology, 49*(10), 1444-1448.

Carroll, D., Ginty, A. T., Painter, R. C., Roseboom, T. J., Phillips, A. C., & de Rooij, S. R. (2012b). Systolic blood pressure reactions to acute stress are associated with future hypertension status in the Dutch Famine Birth Cohort Study. *International Journal of Psychophysiology, 85*(2), 270-273.

Carroll, D., Phillips, A. C., & Der, G. (2008). Body mass index, abdominal adiposity, obesity, and cardiovascular reactions to psychological stress in a large community sample. *Psychosomatic Medicine, 70*(6), 653-660.

Coyne, J. C., Jaarsma, T., Luttik, M.-L., van Sonderen, E., van Veldhuisen, D. J., & Sanderman, R. (2011). Lack of prognostic value of type D personality for mortality in a large sample of heart failure patients. *Psychosomatic Medicine, 73*(7), 557-562.

Cruickshank, J., Neil-Dwyer, G., Dorrance, D., Hayes, Y., & Patel, S. (1989). Acute effects of smoking on blood pressure and cerebral blood flow. *Journal of human hypertension, 3*(6), 443-449.

Denollet, J. (2005). DS14: standard assessment of negative affectivity, social inhibition, and Type D personality. *Psychosomatic Medicine, 67*(1), 89-97.

Denollet, J., Pedersen, S. S., Vrints, C. J., & Conraads, V. M. (2006). Usefulness of type D personality in predicting five-year cardiac events above and beyond concurrent symptoms of stress in patients with coronary heart disease. *American Journal of Cardiology, 97*(7), 970-973.

Denollet, J., Rombouts, H., Gillebert, T., Brutsaert, D., Sys, S., & Stroobant, N. (1996). Personality as independent predictor of long-term mortality in patients with coronary heart disease. *The Lancet, 347*(8999), 417-421.

Denollet, J., Vaes, J., & Brutsaert, D. L. (2000). Inadequate response to treatment in coronary heart disease: adverse effects of type D personality and younger age on 5-year prognosis and quality of life. *Circulation, 102*(6), 630-635.

Ferguson, E., Williams, L., O'Connor, R. C., Howard, S., Hughes, B. M., Johnston, D. W., et al. (2009). A taxometric analysis of type-D personality. *Psychosomatic Medicine, 71*(9), 981-986.

Gallagher, S., Meaney, S., & Muldoon, O. T. (2014). Social identity influences stress appraisals and cardiovascular reactions to acute stress exposure. *British journal of health psychology, 19*(3), 566-579.

Gallagher, S., O'Riordan, A., McMahon, G., & Creaven, A.-M. (2018). Evaluating personality as a moderator of the association between life events stress and cardiovascular reactivity to acute stress. *International Journal of Psychophysiology, 126*, 52-59.

Georgiades, A., Lemne, C., De Faire, U., Lindvall, K., & Fredrikson, M. (1997). Stress-induced blood pressure measurements predict left ventricular mass over three years among borderline hypertensive men. *European journal of clinical investigation, 27*(9), 733-739.

Ginty, A. T., Phillips, A. C., Roseboom, T. J., Carroll, D., & Derooij, S. R. (2012). Cardiovascular and cortisol reactions to acute psychological stress and cognitive ability in the Dutch Famine Birth Cohort Study. *Psychophysiology, 49*(3), 391-400.

Ginty, A. T., Williams, S. E., Jones, A., Roseboom, T. J., Phillips, A. C., Painter, R. C., et al. (2016). Diminished heart rate reactivity to acute psychological stress is associated with enhanced carotid intima-media thickness through adverse health behaviors. *Psychophysiology, 53*(6), 769-775.

Gordan, R., Gwathmey, J. K., & Xie, L.-H. (2015). Autonomic and endocrine control of cardiovascular function. *World journal of cardiology, 7*(4), 204.

Grande, G., Romppel, M., & Barth, J. (2012). Association between type D personality and prognosis in patients with cardiovascular diseases: a systematic review and meta-analysis. *Annals of behavioral medicine, 43*(3), 299-310.

Grande, G., Romppel, M., Vesper, J.-M., Schubmann, R., Glaesmer, H., & Herrmann-Lingen, C. (2011). Type D personality and all-cause mortality in cardiac patients-data from a German cohort study. *Psychosomatic Medicine, 73*(7), 548-556.

Gronwall, D. (1977). Paced auditory serial-addition task: a measure of recovery from concussion. *Perceptual and motor skills, 44*(2), 367-373.

Grynberg, D., Gidron, Y., Denollet, J., & Luminet, O. (2012). Evidence for a cognitive bias of interpretation toward threat in individuals with a Type D personality. *Journal of behavioral medicine, 35*(1), 95-102.

Guelen, I., Westerhof, B. E., van der Sar, G. L., van Montfrans, G. A., Kiemeneij, F., Wesseling, K. H., et al. (2003). Finometer, finger pressure measurements with the possibility to reconstruct brachial pressure. *Blood pressure monitoring, 8*(1), 27-30.

Habra, M. E., Linden, W., Anderson, J. C., & Weinberg, J. (2003). Type D personality is related to cardiovascular and neuroendocrine reactivity to acute stress. *Journal of psychosomatic research, 55*(3), 235-245.

Hartley, T. R., Sung, B. H., Pincomb, G. A., Whitsett, T. L., Wilson, M. F., & Lovallo, W. R. (2000). Hypertension risk status and effect of caffeine on blood pressure. *Hypertension, 36*(1), 137-141.

Howard, S., & Hughes, B. M. (2013). Type D personality is associated with a sensitized cardiovascular response to recurrent stress in men. *Biological psychology, 94*(2), 450-455.

Howard, S., Hughes, B. M., & James, J. E. (2011). Type D personality and hemodynamic reactivity to laboratory stress in women. *International Journal of Psychophysiology, 80*(2), 96-102.

Howard, S., O’Riordan, A., & Nolan, M. (2018). Cognitive Bias of Interpretation in Type D Personality: Associations with Physiological Indices of Arousal. *Applied psychophysiology and biofeedback*, 1-9.

James, J. E., & Richardson, M. (1991). Pressor effects of caffeine and cigarette smoking. *British Journal of Clinical Psychology, 30*(3), 276-278.

Jennings, J. R., Kamarck, T., Stewart, C., Eddy, M., & Johnson, P. (1992). Alternate cardiovascular baseline assessment techniques: Vanilla or resting baseline. *Psychophysiology, 29*(6), 742-750.

Kelly-Hughes, D. H., Wetherell, M. A., & Smith, M. A. (2014). Type D personality and cardiovascular reactivity to an ecologically valid multitasking stressor. *Psychology & health, 29*(10), 1156-1175.

Kupper, N., Denollet, J., Widdershoven, J., & Kop, W. J. (2013a). Type D personality is associated with low cardiovascular reactivity to acute mental stress in heart failure patients. *International Journal of Psychophysiology, 90*(1), 44-49, doi:10.1016/j.ijpsycho.2013.01.011.

Kupper, N., Denollet, J., Widdershoven, J., & Kop, W. J. (2015). Cardiovascular reactivity to mental stress and mortality in patients with heart failure. *JACC: Heart Failure, 3*(5), 373-382.

Kupper, N., Pelle, A., & Denollet, J. (2013b). Association of Type D personality with the autonomic and hemodynamic response to the cold pressor test. *Psychophysiology, 50*(12), 1194-1201.

Linden, W., Frankish, J., & McEachern, H. M. (1985). The effect of noise interference, type of cognitive stessor, and order of task on cardiovascular activity. *International Journal of Psychophysiology, 3*(1), 67-74.

Lovallo, W. R. (2011). Do low levels of stress reactivity signal poor states of health? *Biological psychology, 86*(2), 121-128.

Markovitz, J. H., Raczynski, J. M., Wallace, D., Chettur, V., & Chesney, M. A. (1998). Cardiovascular reactivity to video game predicts subsequent blood pressure increases in young men: The CARDIA study. *Psychosomatic Medicine, 60*(2), 186-191.

Martens, E. J., Mols, F., Burg, M. M., & Denollet, J. (2010). Type D personality predicts clinical events after myocardial infarction, above and beyond disease severity and depression. *The Journal of clinical psychiatry*.

Matthews, K. A., Owens, J. F., Kuller, L. H., Sutton-Tyrrell, K., Lassila, H. C., & Wolfson, S. K. (1998). Stress-induced pulse pressure change predicts women's carotid atherosclerosis. *Stroke, 29*(8), 1525-1530.

Meyer, T., Hussein, S., Lange, H. W., & Herrmann-Lingen, C. (2014). Type D personality is unrelated to major adverse cardiovascular events in patients with coronary artery disease treated by intracoronary stenting. *Annals of Behavioral Medicine, 48*(2), 156-162.

Murdison, K. A., Treiber, F. A., Mensah, G., Davis, H., Thompson, W., & Strong, W. B. (1998). Prediction of Left Ventricular Mass in Youth with Family Histories ofEssential Hypertension. *The American journal of the medical sciences, 315*(2), 118-123.

O'Leary, É. D., Howard, S., Hughes, B. M., & James, J. E. (2013). An experimental test of blunting using sleep-restriction as an acute stressor in Type D and non-Type D women. *International Journal of Psychophysiology, 90*(1), 37-43.

Obrist, P. (1981). Cardiovascular psychophysiolopy. New York: Plenum Press.

Pelle, A. J., Pedersen, S. S., Schiffer, A. A., Szabó, B., Widdershoven, J. W., & Denollet, J. (2010). Psychological Distress and Mortality in Systolic Heart FailureCLINICAL PERSPECTIVE. *Circulation: Heart Failure, 3*(2), 261-267.

Penaz, J. Photoelectric measurement of blood pressure, volume and flow in the finger. In *Digest of the 10th international conference on medical and biological engineering-Dresden, 1973, 1973* (Vol. 104)

Phillips, A. C., Gallagher, S., & Carroll, D. (2009). Social support, social intimacy, and cardiovascular reactions to acute psychological stress. *Annals of Behavioral Medicine, 37*(1), 38-45.

Potter, J. F., Watson, R., Skan, W., & Beevers, D. G. (1986). The pressor and metabolic effects of alcohol in normotensive subjects. *Hypertension, 8*(7), 625-631.

Savoca, M. R., MacKey, M. L., Evans, C. D., Wilson, M., Ludwig, D. A., & Harshfield, G. A. (2005). Association of ambulatory blood pressure and dietary caffeine in adolescents. *American journal of hypertension, 18*(1), 116-120.

Schiffer, A. A., Pedersen, S. S., Widdershoven, J. W., & Denollet, J. (2008). Type D personality and depressive symptoms are independent predictors of impaired health status in chronic heart failure. *European journal of heart failure, 10*(8), 802-810.

Schiffer, A. A., Smith, O. R., Pedersen, S. S., Widdershoven, J. W., & Denollet, J. (2010). Type D personality and cardiac mortality in patients with chronic heart failure. *International journal of cardiology, 142*(3), 230-235.

Schutte, A., Huisman, H., Van Rooyen, J., Malan, N., & Schutte, R. (2004). Validation of the Finometer device for measurement of blood pressure in black women. *Journal of human hypertension, 18*(2), 79.

Schutte, A. E., Huisman, H. W., van Rooyen, J. M., Oosthuizen, W., & Jerling, J. C. (2003). Sensitivity of the Finometer device in detecting acute and medium-term changes in cardiovascular function. *Blood pressure monitoring, 8*(5), 195-201.

Sher, L. (2005). Type D personality: the heart, stress, and cortisol. *Qjm, 98*(5), 323-329.

Somers, V. K., Conway, J., Coats, A., Isea, J., & Sleight, P. (1991). Postexercise hypotension is not sustained in normal and hypertensive humans. *Hypertension, 18*(2), 211-215.

Steptoe, A., & Brydon, L. (2009). Emotional triggering of cardiac events. *Neuroscience & Biobehavioral Reviews, 33*(2), 63-70.

Tadic, M., Cuspidi, C., & Grassi, G. (2018). Heart rate as a predictor of cardiovascular risk. *European journal of clinical investigation, 48*(3), e12892.

Wang, A., Liu, X., Guo, X., Dong, Y., Wu, Y., Huang, Z., et al. (2014). Resting heart rate and risk of hypertension: results of the Kailuan cohort study. *Journal of hypertension, 32*(8), 1600-1605.

Williams, L., O'Connor, R. C., Howard, S., Hughes, B. M., Johnston, D. W., Hay, J. L., et al. (2008). Type-D personality mechanisms of effect: the role of health-related behavior and social support. *Journal of psychosomatic research, 64*(1), 63-69.

Williams, L., O’Carroll, R. E., & O’Connor, R. C. (2009). Type D personality and cardiac output in response to stress. *Psychology and Health, 24*(5), 489-500.

Williams, L., O’Connor, R. C., Grubb, N., & O’Carroll, R. (2011). Type D personality predicts poor medication adherence in myocardial infarction patients. *Psychology & health, 26*(6), 703-712.

Table 1. Means (and standard deviations) for cardiovascular measures across experimental conditions

		Non-social								Social							
		Non-Type D				Type D				Non-Type D				Type D			
		Male		Female		Male		Female		Male		Female		Male		Female	
		M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
DBP																	
	Baseline	73.50	5.95	77.36	9.52	76.47	5.97	78.18	8.3	76.21	7.61	74.24	8.05	71.70	5.58	72.50	5.85
	Task	84.57	9.94	91.59	11.60	83.37	5.47	87.54	12.25	83.28	9.08	85.81	9.05	85.05	9.61	84.72	6.29
SBP																	
	Baseline	124.37	9.87	123.70	15.66	131.31	11.70	123.58	9.70	128.25	13.97	123.18	9.26	127.01	12.23	120.05	7.99
	Task	146.72	19.74	142.78	16.80	145.35	10.18	137.79	18.40	138.86	12.22	143.34	12.82	153.15	26.19	137.05	14.86
HR																	
	Baseline	67.11	7.64	81.36	8.65	84.31	13.11	82.81	9.81	75.03	7.72	80.41	12.39	83.47	9.57	83.48	6.16
	Task	71.76	11.94	91.06	9.20	87.05	13.18	88.79	9.80	76.83	6.94	86.98	16.71	89.54	9.01	85.50	11.11

Note 1. Type D ≥ 10 on both NA and SI subscale for illustrative purposes

Table 2. Means, ranges and standard deviations for cardiovascular reactivity variables

Cardiovascular reactivity variables	Range	Mean	SD
SBP Reactivity	-7.65 - 49.20	18.05	11.20
DBP Reactivity	-3.46 - 25.28	10.66	6.07
HR Reactivity	-7.87- 20.08	5.35	5.36

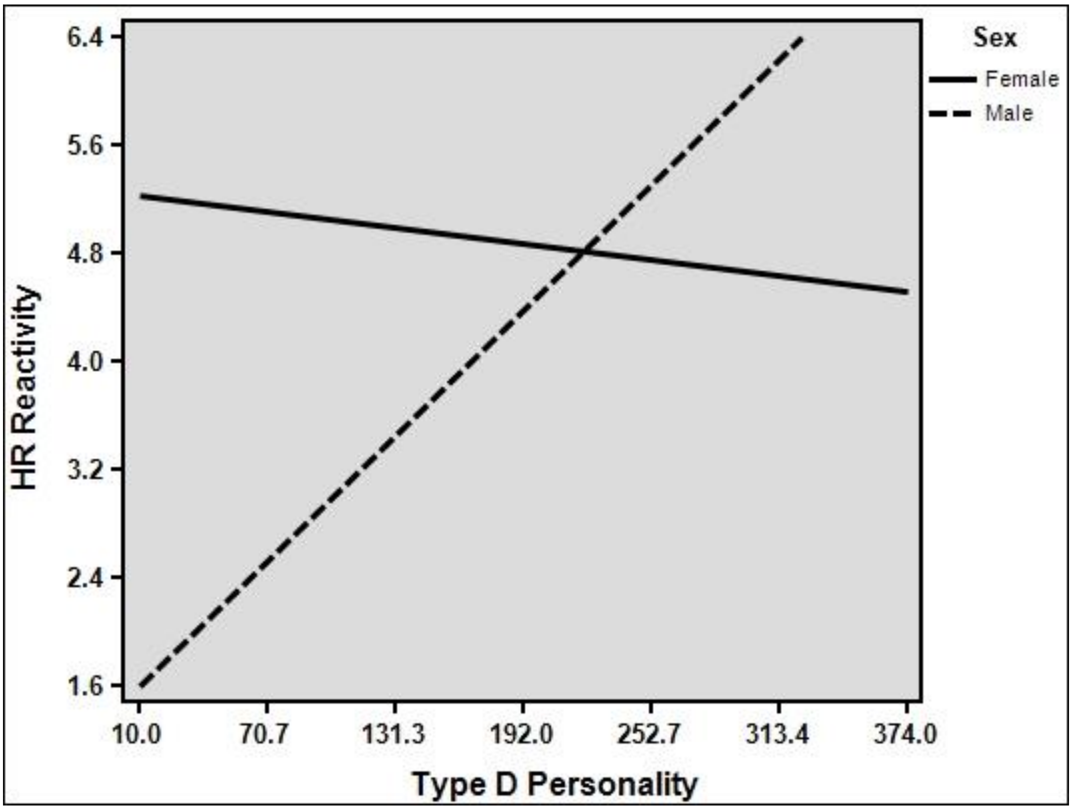

Figure 1. Interaction between Type D personality and sex on HR reactivity in the *Social Condition* (p = .003). Type D was computed as the product of the SI and NA subscales (SIxNA).